REMARKS

Amendments

New claims 118 and 119 define the subject matter cancelled from claims 93 and 109, respectively.

Claims 74, 81 and 87 have been amended to include pharmaceutically acceptable salts of the compounds recited therein. Support for this subject matter is found on page 2, line 28; page 6, lines 13-29 and page 13, lines 29-30; of the specification. This amendment does not raise any new issues in that pending claims 106, 107. 108, and 117 are already directed to pharmaceutically acceptable salts (tosylate).

Claims 74, 81, 87, 106-108 and 117 have been amended to change format errors in the chemical names recited in the claims. The second nitrogen on the urea compound was identified as "**N=**" instead of " **N'**." These claims have also been amended to insert the structural formulas of the compounds defined by chemical names. Support for these formulas is found in examples 42 and 43 of the specification.

Claims 105 and 116 have been cancelled so that all claims are directed to the use of N-(4-chloro-3-(trifluoromethyl)phenyl)-N'-(4-(2-(N-methylcarbamoyl)-4-pyridyloxy)phenyl) urea , N-(4-chloro-3-(trifluoromethyl)phenyl)-N'-(4-(2-carbamoyl-4-pyridyloxy)phenyl) urea or a pharmaceutically acceptable salt of either. The inventorship for all pending claims is the same and comprises each of the inventors identified in the Request To Correct Inventorship filed on October 30, 2006, who are as follows: Bernd Riedl, Jacques Dumas, Uday Khire, Timothy B. Lowinger, William J. Scott, Roger A. Smith, Jill E. Wood and Reina Natero.

Rejections under 35 USC §112

The examiner has yet to present one shred of evidence,which remotely suggests the methods claimed herein are not enabled. In the absence of such evidence, the rejection is deficient under controlling case law. The burden is upon the Patent and Trademark Office to provide evidence shedding doubt that the invention can not be made and used as stated; see for example, *In re Marzocchi*, 439, F. 2d 220, 169 USPQ 367 (CCPA 1971).

The Examiner also has yet to present any evidence which refutes the findings and conclusions made by Kolch et al. (*Nature* **1991**, *349*, 426-28) and Monia et al.,(*Nat. Med.* **1996**, *2*, 668-75), cited in the application, which disclose that raf inhibition is correlated with the inhibition of growth of a variety of tumor types. The examiner instead relies on general conclusions and argues these publications are deficient, stating these publications "do not establish a therapeutic method for the treatment of all types of diseases mediated by RAF kinase generally." The absence of such a disclosure within these publications is not evidence the treatment methods claimed herein are not enabled and does not support the rejections of the methods claimed under 35 USC§ 112. In addition, the absence of such a disclosure within these publications does not diminish the significance of the teachings therein. The data within these publications is sufficient to show that there is a correlation between raf inhibition and the inhibition of growth of a variety of tumor types, refuting the examiner's position. Furthermore, although a therapeutic method for the treatment of all types of diseases mediated by RAF kinase generally is not expressly disclosed by Kloch et al or Monia et al., such a method, were its disclosure necessary for enablement, which is not the case, is suggested by Monia et al, as evidenced by the following statements:

> These studies strongly suggest that antisense inhibitors targeted against C-raf -1 kinase may be of considerable value as antineoplastic agents that display activity against a wide spectrum of tumor types at well-tolerated doses. Monia et al. Abstract, p 668

> The ability to use antisense ODNs to target selectively the genetic processes involved in cancer has raised the exciting possibility that these compounds could be used, not only as a new class of chemotherapeutic agent, but also to gain a better understanding of the critical molecular events responsible for

initiating and maintaining the cancer phenotype. Monia et al. p. 673, col. 2, lines 5-10.

> The recent discovery that raf kinases function in part as downstream mediators of ras oncogene action suggests that inhibitors of raf gene expression may prove useful in the treatment of ras-dependent tumors. Monia et al. p.673, col. 2, lines 17-21.

Monia et al. also expressly refers to "ISIS 5132", a pharmaceutical agent which is a c-raf inhibitor, and suggests it is effective against a broad range of diseases, in the following lines:

> Thus, studies examining the effects of ISIS 5132 against a broader spectrum of tumor types and characterization of the anti-tumor properties of antisense inhibitors designed against other raf kinase family members and other members of the MAP kinase signaling pathway, both alone and in combination, are of obvious importance. Monia et al. p 673, col. 2, line 57-62.

Monia presented additional data in a paper for the 1997 Ciba Foundation Symposium 209, Oligonucleotides as Therapeutic Agent, Wiley, Chichester, p 107-123, showing that the raf kinase inhibitor ISIS 5132 exhibited anti-tumor activity in nude mouse tumor xenografts with the following varying tumor types: lung (3 types), prostate (2 types), bladder, breast (2 types), melanoma (2 types), colon (3 types) and small-cell lung (2 types); see Table 2, page 112. This data reinforced the teachings in the Monia et al. (1996) publication that raf inhibition is correlated with the inhibition of growth of a variety of tumor types.

Following the 1996 publication by Monia et al., the raf kinase inhibitor ISIS 5132 was in fact administered to patients with various diseases in clinical trials. The following are reported on the National Cancer Institute Web site "Clinical trials (PDQ®)":

a) Phase II Radomized Study of ISIS 5132 or ISIS 3521 in Women with Previously Treated Metastatic Breast Cancer(first published 4/1/1998, last modified 3/2/2004)

b) Phase II Radomized Study of ISIS 5132 and ISIS 3521 for Locally Advanced or Metastatic Colorectal Cancer(first published 8/1/1998, last modified 12/1/1999)

c) Phase II Radomized Study of ISIS 5132 and ISIS 3521 in Patients with Hormone Refractory Prostate Cancer (first published 8/1/1998, last modified 7/1/1999) and

d) Phase II Study of ISIS 5132 in Patients with Advanced Pancreatic Cancer (first published 5/1/1998, last modified 8/1/1999).

See also, www.NewRx.com, Purchased Articles Antisense Technology Phase II Trial of Second Antisense Cancer Drug Begins.

In addition to the disclosures within these abstracts, the following publications disclose the administration of the raf kinase inhibitor ISIS 5132 and ISIS 3521 to patients with renal cancer, colon cancer, melanoma, lymphoma, ovarian cancer, non-small cell lung cancer, small cell lung cancer, mesothelioma, colorectal cancer and sarcoma.

Phase I Trial of C-raf Antisense Oligonucletide ISIS 5132 (CGP 69846A) By 21-day Continuous Intravenous Infusion(CIV) in Patients with Advanced Cancer , ASCO Abstract (1998) .

Phase I Clinical/Pharmacokinetic and Pharmacodynamic trial of the c-raf-1Antisense Oligonucleotide ISIS 5132(CGP69846A) Journal of Clinical Oncology, Vol. 17, No.7 (July) 1999: pp 2227-2236. Submitted November 5, 1998.

Phase I Evaluation of ISIS 3521, an Antisense Oiligonacleotide to Protein Kinase C-alpha, in Patients with Advanced Cancer. Journal of Clinical Oncology, Vol 17, No. 11 (Nov) 1999, pp. 3586-3595.

These publications illustrate that the treatment of various diseases with a raf kinase inhibitor, as suggested by Monia et al. (1996), was considered more than speculative at the time this application was filed. Those skilled in the art recognized that the raf kinase inhibitor ISIS 5132 had sufficient utility to be administered to patents with a wide variety of tumor types.

In addition to the teachings relating to the raf kinase inhibitor ISIS 5132, other raf kinase inhibitors known in the art, such as benzamides and azaquinoxaline compounds, were described as effective against a variety of diseases.

a) Benzamides

WO 98/22103 discloses benzamide raf kinase inhibitors which are said to be "especially useful in treatment of tumours having a high incidence of ras mutation, such as colon, lung, and pancreatic tumors." See page 15, lines 10-13. On page 2, lines 10-22, it is stated, "inhibition of the kinase activity of raf is expected to have antitumour activity in a least a portion of human tumours."

Cancers of interest are said to include, "carcinoma, including that of the bladder, breast, colon, kidney, liver, lung, ovary, pancreas, stomach, cervix, thyroid and skin;

hematopoietic tumors of lymphoid lineage, including acute lymphocytic leukemia, B-cell lymphoma and Burkett's lymphoma;

hematopoietic tumors of myeloid lineage, including acute and chronic myelogenous leukemias and promyelocytic-leukemia;

tumors of mesenchymal origin, including fibrosis or coma and rhabdomyosarcoma; and

other tumors, including melanoma, seminoma, tetratocarcinoma, neuroblastoma and glioma."

b) Azaquinoxalines

US Patent No. 6,204,267, filed May 1, 1998, and US Patent No. 6,180,631, filed October 5, 1998, disclose and claim the treatment of lung cancer, ovarian cancer, breast cancer, brain cancer, intra-axial brain cancer, colon cancer, prostate cancer, Kaposi's sarcoma, melanoma, and glioma with azaquinoxaline compounds said to modulate the function of serine/threonine protein kinases, including raf. (see claim 18 of '267 and claim 3 of '631).

Based on these disclosures, one skilled in the art would recognize that applicants' claims to treating multiple diseases are not unusual.

Other agents

As shown by the 18 patents cited in the previous reply, there are also agents other than raf kinase inhibitors which are described by those skilled in the art as effective for the treatment of a variety of disorders (carcinomas, myeloid disorders and adenomas) or tumors.

The examiner has not presented any evidence to refute the disclosures made in these patents. The examiner instead states that these patents "do not conclusively provide to one of ordinary skill in the art that the compounds disclosed therein would be effective in the treatment of all types of solid tumors, carcinomas, myeloid disorders or adenomas" and takes the position all are invalid without providing any supporting evidence. The mentioned 18 patents are those found in reviewing only a portion of the patents uncovered in a key word search. A search for the phrase "solid tumor" in the claims of US patents resulted in 450 hits. The 18 patents show at least that the asserted utility in treating various disorders such as solid tumors, carcinoma, myeloid disorder or adenoma with a single agent is not "revolutionary" and whether validly issued or not, these patents demonstrate that the examiner's general conclusions and assumptions that the "the state of the art does not identify a single class of compounds that can treat all these types of cancers generally" are erroneous.

On page 3, line 15, to page 4, line 3, of the office action the examiner defends the absence of evidence to support the rejection in stating that the claims are drawn to several types of cancers affecting different organs and having different methods of growth or harm to the body. The examiner states that the carcinogenic process is a multi-step multi-mechanism process and that no two cancers are alike, concluding that a single therapeutic approach does not exist. Applicants respectfully request the Examiner provide a declaration with these statements and conclusions in that they are not supported by evidence and they are inconsistent with the teachings in the prior art references of record such as Monia et al (1996) and the 18 patents mentioned in the previous reply. In addition, despite the examiner's analysis, a number of pharmaceutical agents been approved by the FDA for the treatment of more than one type of cancer. Examples of these agents include docetaxel (non-small cell lung cancer, prostate cancer, breast cancer, head and neck cancer) gemcitabine (pancreatic cancer, non-small cell lung cancer, ovarian cancer, breast cancer) and paclitaxel (ovarian cancer, breast cancer, non-small cell lung cancer). These facts contradict the Examiner's conclusion.

Although the examiner provides no basis or evidence that the disclosed assays are insufficient to establish the activity of the recited compounds as raf kinase inhibitors or that there is reason to doubt the correlation between the results of raf kinase assays employed and clinical efficacy for treatment of the claimed diseases, the Examiner requires a reference which establishes such a correlation. This requirement ignores the correlation of raf kinase inhibition and the treatment of various conditions taught by Monia et al (1996), which alone is sufficient to provide the evidence the examiner requires. Other publications by Monia , Kloch et al. Daum et al. and Fridman et al provide evidence of this correlation. Furthermore, the treatment of the claimed diseases is not "unusual, difficult or speculative" so as to require such evidence. There are many chemical agents, e.g., docetaxel, gemcitabine and paclitaxel, which have been approved for treating solid cancers in a human.

Contrary to the examiner's allegations, Applicants maintain that the express disclosure within the specification is sufficient to enable the claims herein and that further assays or data to support the methods of treatment are not necessary. Based on the teachings within the art of the broad spectrum of activity of raf kinase inhibitors, one skilled in the art would recognize that the compounds recited in the claims herein, having raf kinase activity, would be effective in treating a variety of diseases. Therefore, there clearly is no need for any assays or data, let alone additional assays or data to support the claims herein. No evidence has been presented to the contrary.

Applicants clearly provide sufficient guidance to make and use the invention. As discussed above, the synthesis of the two recited compounds is described on page 66. Methods for preparing pharmaceutical compositions with these compounds and methods for administering compounds in the treatment of cancers are provided on pages 10-14. Dosages are provided on page 13, lines 11-20. To the extent the disclosure does not provide specific dosages, it would at most involve routine experimentation, if any at all, for one skilled in the art to treat any one of the recited cancers with the compounds of this invention. The enablement requirement is satisfied if, "the specification teaches those in the art enough that

they can make and use the claimed invention without "undue experimentation" See, *Amgen Inc. v. Hoechst Marion Roussel,* 314 F.2d 1313, 65 USPQ 2nd 1385 (Fed Cir. 2003). Using the claimed compounds would be routine for those skilled in the art in view of Applicant's disclosure.

The examiner claims Applicants' reliance on the decision in *In re Brana,* 51 F.3d 1560, 34 USPQ2d 1436 (Fed. Cir. 1995) is erroneous based on alleged differences in the facts. Applicants maintain their reliance on *In re Brana* is appropriate in that they relied on this citation for the general legal principle that an applicant is not required to test the claimed invention in its final use. This case is not relied on for the underlying facts. Furthermore, Applicants' maintain the alleged differences in the underlining facts either do not exist or are not relevant to the decision in *Brana.*

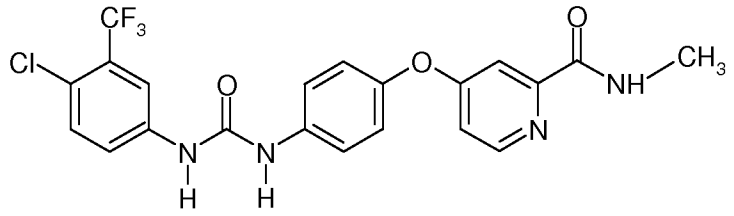
The examiner finds that in *Brana*, the compounds on appeal were narrower in scope than those herein. Applicants respectfully disagree. The method claims herein recite 2 compounds and their pharmaceutically acceptable salts. In contrast, the compounds of *Brana* were defined by a formula with 4 variable substituents, each defined by a Markush group comprising over 10 different moieties. The claims herein are clearly narrower in scope than the claims in *Brana*.

While the invention at issue in *Brana* was to a pharmaceutical compound and not a method of treatment, there is no indication that a) the relied on aspects of the decision were restricted to inventions relating to compositions of matter or 2) methods of treatment should be held to such a different standard in satisfying the disclosure requirements under 35 USC§112 as to require actual data showing end use, i.e., sufficient for FDA approval, to satisfy the statute. Such a distinction would be inconsistent with the Court's reasoning since such a strict disclosure requirement to obtain a method claim would discourage innovation, the same as it would if necessary to obtain a claim to a composition of matter.

The nature of the in-vivo tests being art recognized was relevant in *Brana* since the

specification did not describe the details of what the assay comprised and did not disclose objective values obtained from the assay with the claimed compounds. The specification merely disclosed a relative comparison of the compounds claimed to other compounds known in the art through this assay. Since the specification herein describes the assay performed and objective values (within a range) obtained from an assay of the compounds recited in the claims, the assay employed need not be art recognized at the time the invention is made for the application to be enabling.

FDA Approval



The captioned compound (known as Sorafenib or Nexavar®) was approved by the FDA for treatment of renal carcinoma. In addition, it has been used in clinical trials (alone and in combination) for the treatment of other cancers such as melanoma, non-small cell lung cancer and heptacellular carcinoma.

This clearly shows its efficacy in testing various types of cancer was so good in *in vitro* and *in vivo* assays correlated with such cancers that it warranted the expense of clinical trials sanctioned by the FDA. Were such a showing of efficacy against a spectrum of cancers needed for enablement (it is not), these clinical trials provide it, i.e., they provide sufficient evidence of efficacy for a wide spectrum of tumors which is more than adequate for patent purposes.

Not all clinical trials for Sorafenib have resulted in approvals for treatment and some clinical trials were stopped (melanoma) for insufficient efficacy. These clinical trials are for determining efficacy and safety, which is beyond what is necessary to satisfy the enablement

requirement of 35 USC §112, first paragraph. As stated in *In re Brana*, 51 F.3d 1560, 34 USPQ2d 1436, 1442, (Fed. Cir. 1995) with respect to the utility requirement,

> FDA approval, however, is not a prerequisite for finding a compound useful within the meaning of the patent laws. *Scott*, 34 F.3d 1058, 1063, 32 USPQ2d 1115, 1120. Usefulness in patent law, and in particular in the context of pharmaceutical inventions, necessarily includes the expectation of further research and development. The stage at which an invention in this field becomes useful is well before it is ready to be administered to humans. Were we to require Phase II testing in order to prove utility, the associated costs will prevent any companies from obtaining patent protection on the promising new invention, thereby eliminating an incentive to pursue, through research and development, potential cures in any crucial area such as the treatment of cancer.

As stated in *In re Anthony*, 414 F2d 1383, 162 USPQ 594, 604 (CCPA 1969), "Approval by the FDA, is not a prerequisite for the patenting of a new drug." As to the issue of safety, *In re Anthony* held,

> ...Congress has given the responsibility to the FDA, not the Patent Office, to determine in the first instance whether drugs are sufficiently safe for use that they can be introduced in the commercial market, under the conditions prescribed, recommended or suggested in the proposed labeling thereof, as the majority of this court noted in *Hartop*, 135 USPQ at 426, 427.

Only measurable efficacy is required to satisfy the statute.

Thus, even under the examiner's incorrect standard for enablement, the claims satisfy 35 U.S.C. §112.

Dependent claims

The Examiner has not provided an appropriate analysis of the scope of the certain dependent claims herein in rejecting them under 35 U.S.C. §112, first paragraph. The Examiner has collectively analyzed all claims as having similar scope and has assumed the claims embrace the treatment of all types of solid tumors, carcinomas, myeloid disorders and adenomas. For example, the examiner's reasoning to support the rejection under 35 USC 112, first paragraph, is that "the state of the art references do not establish a therapeutic method

for the treatment of all types of diseases mediated by RAF kinase generally." This reasoning does not apply to dependent claims where only one condition is specified. (Claims 93, 100-104, 109, 111-115, 118 and 119.) In that the dependent claims do not suffer from the alleged deficiencies, the rejection of these claims should be withdrawn.

Claim 117

The examiner alleges the specification does not enable claim 117, which is directed to a method for inhibiting RAF-kinase in a human or mammal comprising administering a tosylate salt of *N*-(4-chloro-3-(trifluoromethyl) phenyl)-*N'*-(4-(2-carbamoyl-4-pyridyloxy) phenyl) urea or *N*-(4-chloro-3-(trifluoromethyl) phenyl)-*N'*-(4-(2-(*N*-methylcarbamoyl)-4-pyridyloxy) phenyl) urea. The specification provides sufficient guidance to prepare the two urea compounds and also provides sufficient guidance on how to prepare and administer compositions with these compounds, including dosages. The specification also shows that the free base of these compounds, compounds 42 and 43, inhibit raf kinase in the assays disclosed.

The examiner has not identified any element of the claim for which the disclosure is allegedly deficient and has not identified any claim term, which is allegedly indefinite. Instead, the examiner reads limitations into the claim regarding the treatment of diseases. Incorporating limitations into a claim is improper, *See Phillips v. AWH Corp.,* 415 F.3d 1303, 1313, 75 USPQ2d 1321, 1326 (Fed. Cir. 2005) (en banc) and inconsistent with the MPEP. MPEP 2111.01 states "words of the claim must be given their plain meaning unless the plain meaning is inconsistent with the specification" (*See In re Zletz*, 893 F.2d 319, 321, 13 USPQ2d 1320, 1322 (Fed. Cir. 1989)) and "it is important not to import into a claim limitations that are not part of the claim."

In that there is no basis for referring to the specification for the meaning of any claim term and there clearly is no basis for reading treatment limitations into the claim, the rejection of claim 117 should be withdrawn for these reasons alone.

The examiner alleges the claim does not satisfy the statute on the basis that, "applicant did not state on the record or provide any guidance that the assays provided are correlated to the clinical efficacy of the treatment of various disorders intended by the instant claim." This is not a proper basis for rejecting the claim in that it improperly requires incorporation of treatment limitations, as discussed above. In addition, such a showing is not necessary here. The specification provides an objectively enabling disclosure and there is no necessity for any data at all. In any event,
this reasoning is defective in that applicants have cited publications and facts which have correlated raf kinase activity with the treatment of various diseases. While these publications describe different assay techniques for measuring raf kinase activity, there is no basis to question the accuracy of the assays used in determining the inhibition of raf kinase activity. The applicants were employed by the assignee, a pharmaceutical manufacturer, at the time of their invention, which would only use assays that were reasonably correlated with efficacy to find new products. No evidence has been presented to the contrary.

The examiner indicates that in-vitro activity data holds a "significant role in determining the dosage regimen." Applicants submit that they have provided sufficient disclosure to determine a dosage regimen and no evidence has been presented the disclosure is deficient in that regard in any way.

The examiner cites the reference Keller et al (Biochemical Pharmacology 2004) The role of Raf kinase inhibitor Protein (RKIP) in Health and Disease (2004) to support the rejection. Keller et al teach that a recently discovered Raf kinase inhibitor protein (RKIP) is a natural protein which has a wide tissue expression in many mammals, has been assigned multiple functions and is associated with an increasing number of diseases. In this article, Keller et al. describe RKIP's molecular role in signaling, its physiological functions and its role in disease and suggests it may be a target for therapeutic interventions.

There is no correlation made between RKIP and the raf protein (RKIP is said to bind

Raf -1) and there is no correlation made between RKIP and conventional raf kinase inhibitors used as therapeutic agents. There is also no suggestion that RKIP be used as a therapeutic agent to treat diseases such as raf mediated diseases. Therefore, this publication has little if any relevance to the claimed invention.

Keller al. do suggest that RKIP is a "molecular target for compounds designed for cancer treatment" and "targets for therapeutic interventions" for other diseases. The "additional studies" called for in the language cited by the examiner is to precisely determine RKIP's role in diseases to further determine if it is a suitable target for therapeutic agents. Such a target is distinct from the raf protein target inhibited by the compounds recited in the claims. Therefore, the teachings therein present no insight into methods of the present invention. Furthermore, other than the call for additional research to identify therapeutic agents (such as those of the present invention), the examiner has not cited any discrepancies between the teachings within Keller et al. and the present invention. The examiner's conclusion that to practice the invention of claim 117 will require undue experimentation is based on evidence of no relevance to the invention claimed and should be withdrawn.

For the reasons indicated above, Applicants maintain that they have provided more than adequate guidance and examples to enable the claimed invention and submit all claims meet the requirements of 35 U.S.C. §112, first and second paragraphs.

Rejections under 35 USC §102

The inventorship for all pending claims is the same and comprises each of the inventors identified in the Request To Correct Inventorship filed on October 30, 2006, who are as follows: Bernd Riedl, Jacques Dumas, Uday Khire, Timothy B. Lowinger, William J. Scott, Roger A. Smith, Jill E. Wood and Reina Natero.

Attached are executed declarations under 37 CFR §132 indicating that those named are the co-inventors of the subject matter defined in the pending claims (claims 74, 81, 87, 93,

99, 100-104, 106-115 and 117-119), that those named invented the subject matter of the pending claims at least as early January 13, 1999, as evidenced by cited disclosures within US PROVISIONAL APPLICATION NUMBER 60/115,877, and that any description of the subject matter of the pending claims of these applications within PCT US 00/00768, filed 01/13/2000 (WO 00/041698), and PCT US 00/00648, filed 01/13/2000(WO 00/042012), is a description of their invention. Thus, the disclosures of applicants' invention within WO 00/041698 and WO 00/042012 can not be relied in rejecting the pending claims under 35 USC§ 102(a), and applicants respectfully maintain this rejection be withdrawn.

Furthermore, all of these applications mentioned in the attached 132 declarations and the inventions disclosed therein have at all times been commonly owned by the assignee herein.

Declarations executed by Bernd Reidl, Jacques Dumas, Tim Lowinger and William Smith are attached hereto. Declarations from the other inventors have not been obtained at this time in that they have relocated and/or sought new employment following the closing of the facility in West Haven, Connecticut by Bayer Pharmaceuticals, Inc.

Obviousness type Double Patenting:

Essentially all pending claims except claim 117 have been rejected under the doctrine of obviousness type double patenting in view of claims 67, 73, 78 and 83 of copending application 10/042,226 and claim 67 of copending application 09/948,915.

To render these rejections moot, claim 67 has been canceled from 09/948, 915 and claims 67, 73, 78 and 83 have been cancelled from copending application 10/042,226. See the attached copies.

Information Disclosure Statement:

An Information Disclosure Statement has been filed with this response making the publications regarding ISIS 5132 and other early raf kinase inhibitors of record. Also of record is a sampling of publications disclosing the use of Sorafenib (Bay 43-9086) in clinical

trials. In view of the above remarks, favorable reconsideration of all rejections is courteously requested. If there are any remaining issues, which could be expedited by a telephone conference, the Examiner is courteously invited to telephone counsel at the number indicated below.

The Commissioner is hereby authorized to charge any fees associated with this response or credit any overpayment to Deposit Account No. 13-3402.

Respectfully submitted,

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